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 NEWS  1           Web Page for STN Seminar Schedule - N. America
 NEWS  2  JUL 02   LMEDLINE coverage updated
 NEWS  3  JUL 02   SCISEARCH enhanced with complete author names
 NEWS  4  JUL 02   CHEMCATS accession numbers revised
 NEWS  5  JUL 02   CA/CAplus enhanced with utility model patents from China
 NEWS  6  JUL 16   CAplus enhanced with French and German abstracts
 NEWS  7  JUL 18   CA/CAplus patent coverage enhanced
 NEWS  8  JUL 26   USPATFULL/USPAT2 enhanced with IPC reclassification
 NEWS  9  JUL 30   USGENE now available on STN
 NEWS 10  AUG 06   CAS REGISTRY enhanced with new experimental property tags
 NEWS 11  AUG 06   BEILSTEIN updated with new compounds
 NEWS 12  AUG 06   FSTA enhanced with new thesaurus edition
 NEWS 13  AUG 13   CA/CAplus enhanced with additional kind codes for granted
                   patents
 NEWS 14  AUG 20   CA/CAplus enhanced with CAS indexing in pre-1907 records
 NEWS 15  AUG 27   Full-text patent databases enhanced with predefined
                   patent family display formats from INPADOCDB
 NEWS 16  AUG 27   USPATOLD now available on STN
 NEWS 17  AUG 28   CAS REGISTRY enhanced with additional experimental
                   spectral property data
 NEWS 18  SEP 07   STN AnaVist, Version 2.0, now available with Derwent
                   World Patents Index
 NEWS 19  SEP 13   FORIS renamed to SOFIS
 NEWS 20  SEP 13   INPADOCDB enhanced with monthly SDI frequency
 NEWS 21  SEP 17   CA/CAplus enhanced with printed CA page images from
                   1967-1998
 NEWS 22  SEP 17   CAplus coverage extended to include traditional medicine
                   patents

 NEWS EXPRESS  19 SEPTEMBER 2007:  CURRENT WINDOWS VERSION IS V8.2,
               CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
               AND CURRENT DISCOVER FILE IS DATED 19 SEPTEMBER 2007.

 NEWS HOURS    STN Operating Hours Plus Help Desk Availability
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 NEWS IPC8     For general information regarding STN implementation of IPC 8

Enter NEWS followed by the item number or name to see news on that
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FILE 'HOME' ENTERED AT 16:54:38 ON 22 SEP 2007

=> file medline, uspatful, dgene, embase, wpids
COST IN U.S. DOLLARS SINCE FILE ENTRY TOTAL SESSION
FULL ESTIMATED COST 0.42 0.42

FILE 'MEDLINE' ENTERED AT 16:55:44 ON 22 SEP 2007

FILE 'USPATFULL' ENTERED AT 16:55:44 ON 22 SEP 2007
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=> s (decarboxylase65)
L1 9 (DECARBOXYLASE65)

=> d l1 ti abs ibib tot

L1 ANSWER 1 OF 9 MEDLINE on STN
TI A role for L-type calcium channels in the maturation of parvalbumin-containing hippocampal interneurons.
AB While inhibitory interneurons are well recognized to play critical roles in the brain, relatively little is know about the molecular events that regulate their growth and differentiation. Calcium ions are thought to be important in neuronal development and L-type voltage gated Ca(+2) channels have been implicated in activity-dependent mechanisms of early-life. However, few studies have examined the role of these channels in the maturation of interneurons. The studies reported here were conducted in hippocampal slice cultures and indicate that the L-type Ca(+2) channel agonists and antagonists accelerate and suppress respectively the growth of parvalbumin-containing interneurons. The effects of channel blockade were reversible suggesting they are not the result of interneuronal cell death. Results from immunoblotting showed that these drugs have similar effects on the expression of the GABA synthetic enzymes, glutamic acid decarboxylase65, glutamic acid decarboxylase67 and the vesicular GABA transporter. This suggests that L-type Ca(+2) channels regulate not only parvalbumin expression but also interneuron development. These effects are likely mediated by actions on the interneurons themselves since the alpha subunits of L-type channels, voltage-gated calcium channel subunit 1.2 and voltage-gated calcium channel subunit 1.3 were found to be highly expressed in neonatal mouse hippocampus and co-localized with parvalbumin in interneurons. Results also showed that while these interneurons can contain either subunit, voltage-gated calcium channel subunit 1.3 was more widely expressed. Taken together results suggest that an important subset of developing interneurons expresses L-type Ca(+2) channels alpha subunits, voltage-gated calcium channel subunit 1.2 and especially voltage-gated calcium channel subunit 1.3 and that these channels likely regulate the development of these interneurons in an activity-dependent manner.
ACCESSION NUMBER: 2005539367 MEDLINE
DOCUMENT NUMBER: PubMed ID: 16154277
TITLE: A role for L-type calcium channels in the maturation of parvalbumin-containing hippocampal interneurons.
AUTHOR: Jiang M; Swann J W
CORPORATE SOURCE: The Cain Foundation Laboratories, Department of Pediatrics, Baylor College of Medicine, 6621 Fannin Street, MC 3-6365,

Houston, TX 77030, USA.
CONTRACT NUMBER: NS18309 (NINDS)
NS37171 (NINDS)
SOURCE: Neuroscience, (2005) Vol. 135, No. 3, pp. 839-50.
Electronic Publication: 2005-09-08.
Journal code: 7605074. ISSN: 0306-4522.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200512
ENTRY DATE: Entered STN: 12 Oct 2005
Last Updated on STN: 23 Dec 2005
Entered Medline: 22 Dec 2005

L1 ANSWER 2 OF 9 MEDLINE on STN
TI Perinatal factors and development of islet autoimmunity in early childhood: the diabetes autoimmunity study in the young.
AB The objective of this study was to test whether maternal age at delivery, child's birth order, cesarean section, complicated delivery, maternal smoking during pregnancy, or neonatal jaundice predict islet autoimmunity in children at genetically increased risk of type 1 diabetes in a birth cohort with blood draws at ages 9, 15, and 24 months and yearly thereafter. Newborns with diabetes-associated human leukocyte antigen genotypes (n = 938) and offspring or siblings of persons with type 1 diabetes (n = 428) from the Denver, Colorado, metropolitan area were examined from January 1994 to February 2003. Information on perinatal factors was collected by using questionnaires soon after the birth. Islet autoimmunity was defined as positivity for > or = 1 autoantibody to glutamic acid decarboxylase65, insulin, or protein tyrosine phosphatase-2/ICA512 at > or = 2 consecutive visits (n = 52; mean follow-up, 3.9 years). Complicated delivery (breech, forceps, vacuum extraction) predicted a higher risk of islet autoimmunity (hazard ratio = 2.10, 95% confidence interval: 1.09, 4.05). Increasing maternal age was related to risk of islet autoimmunity among first-degree relatives of persons with type 1 diabetes (hazard ratios = 3.96 and 8.88 for maternal ages 25-34 and > or = 35 years, respectively, compared with < 25 years; p for trend = 0.008. Other factors evaluated were not related to risk of islet autoimmunity. In conclusion, influences in utero or during delivery may affect the fetal immune system.

ACCESSION NUMBER: 2004326861 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15229111
TITLE: Perinatal factors and development of islet autoimmunity in early childhood: the diabetes autoimmunity study in the young.
AUTHOR: Stene Lars C; Barriga Katherine; Norris Jill M; Hoffman Michelle; Erlich Henry A; Eisenbarth George S; McDuffie Robert S Jr; Rewers Marian
CORPORATE SOURCE: Barbara Davis Center for Childhood Diabetes, University of Colorado Health Sciences Center, Denver, CO 80262, USA.
CONTRACT NUMBER: DK-32083 (NIDDK)
DK-32493 (NIDDK)
P30 DK 57516 (NIDDK)
SOURCE: American journal of epidemiology, (2004 Jul 1) Vol. 160, No. 1, pp. 3-10.
Journal code: 7910653. ISSN: 0002-9262.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English

FILE SEGMENT: Priority Journals
ENTRY MONTH: 200408
ENTRY DATE: Entered STN: 2 Jul 2004
Last Updated on STN: 4 Aug 2004
Entered Medline: 3 Aug 2004

L1 ANSWER 3 OF 9 MEDLINE on STN
TI Distribution of glutamate decarboxylase65 immunoreactive puncta on pyramidal and nonpyramidal neurons in hippocampus of schizophrenic brain.
AB Recent studies have reported an increase in GABAA receptor binding activity in several key corticolimbic regions, including the hippocampal formation, of postmortem schizophrenic brain. Because this change has been postulated to represent a compensatory upregulation of this receptor, the current report has sought to determine whether a decrease of glutamate decarboxylase (GAD), the enzyme responsible for the synthesis of GABA, may also be present in the hippocampus of schizophrenic subjects. A standard immunoperoxidase technique, together with a computer-assisted microscopic analysis, has been employed to evaluate the distribution of the 65 kDalton isoform of GAD (GAD65) in 12 normal controls and 13 schizophrenic subjects matched for age and postmortem interval (PMI). The results show no significant difference in the density of GAD65-immunoreactive (-IR) puncta in contact with pyramidal neurons (PN), nonpyramidal neurons (NP), or neuropil (NPL) in sectors CA1-4 and their various sub-laminae. When the data were considered in relation to neuroleptic exposure, a significant positive correlation between the density of GAD65-IR puncta and drug dose was found on both PNs ($r = 0.814$, $P = 0.002$; $r = 0.777$, $P = 0.005$, respectively) and NPs ($r = 0.673$, $P = 0.023$; $r = 0.672$, $P = 0.024$, respectively) in sectors CA4 and CA3. A similar result was found in the stratum oriens of CA3 ($r = 0.704$, $P = 0.016$) and CA2 ($r = 0.774$, $P = 0.009$). In each instance, two neuroleptic free schizophrenics showed the lowest density of GAD65-IR puncta. There was no significant relationship between the density of GAD65-IR puncta with either age or PMI. Taken together with previous data showing an upregulation of GABAA receptor activity in sectors CA3 and CA2, particularly the stratum oriens, this study provides further evidence in support of the hypothesis that an intrinsic defect of GABAergic activity may occur in the hippocampal formation of schizophrenic patients and show dose-related increases in relation to neuroleptic exposure.
ACCESSION NUMBER: 1998325727 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9661250
TITLE: Distribution of glutamate decarboxylase65 immunoreactive puncta on pyramidal and nonpyramidal neurons in hippocampus of schizophrenic brain.
AUTHOR: Todtenkopf M S; Benes F M
CORPORATE SOURCE: Laboratory for Structural Neuroscience, McLean Hospital, Belmont, Massachusetts 02178, USA.
CONTRACT NUMBER: MH00423 (NIMH)
MH31862 (NIMH)
MH42261 (NIMH)
+
SOURCE: Synapse (New York, N.Y.), (1998 Aug) Vol. 29, No. 4, pp. 323-32.
Journal code: 8806914. ISSN: 0887-4476.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199809
ENTRY DATE: Entered STN: 6 Oct 1998
Last Updated on STN: 6 Oct 1998
Entered Medline: 24 Sep 1998

L1 ANSWER 4 OF 9 MEDLINE on STN

TI Glutamic acid decarboxylase65 (GAD65) antibodies and insulin auto-antibodies in Japanese patients with non-insulin-dependent diabetes mellitus.

AB To clarify whether glutamic acid decarboxylase65 antibodies (GAD65 Ab) and insulin autoantibodies (IAA) are good predictive markers for insulin-dependency in NIDDM, we studied GAD65 Ab and IAA in NIDDM patients treated with diet alone or in combination with oral hypoglycemic agents. GAD65 Ab were found in 12 or 29 (5.2%, P = 0.079 vs. control) NIDDM patients and IAA in 8 of 229 (3.5%). The frequency of GAD65 Ab and IAA positivity in NIDDM did not differ significantly from those of healthy controls (2/150, 1.3%, 2/150, 1.3%, respectively), but the frequency of patients who were positive for either GAD65 Ab or IAA, or both, was significantly higher than that of normal controls (17/229, 7.4% and 4/150, 2.7%, respectively, P < 0.05). In addition, the prevalences of GAD65 Ab and of IAA in those patients whose disease durations, since the diagnosis of diabetes, were less than one year were significantly higher than those of controls (4/30, 13.3%, P < 0.05, 4/30, 13.3%, P < 0.05, respectively). We found no differences between GAD65 Ab positive- and negative-patients in either BMI or serum C-peptide levels. Over a one to five year follow-up period (mean 2.0 yrs), serum C-peptide levels gradually decreased necessitating insulin treatment in three of the patients positive for GAD65 Ab and/or IAA (3/17, 17.6%; two were positive for both GAD65 Ab and IAA and one was positive for GAD65 Ab only). In contrast, only five patients negative for the two antibodies developed insulin requirement (5/212, 2.4%, P < 0.01). These results suggest that GAD65 Ab and IAA are good markers for predicting the development of insulin dependency in NIDDM patients and that the predictive value for insulin-dependency in NIDDM is enhanced by measuring both antibodies.

ACCESSION NUMBER: 97297144 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9152613
TITLE: Glutamic acid decarboxylase65 (GAD65) antibodies and insulin auto-antibodies in Japanese patients with non-insulin-dependent diabetes mellitus.
AUTHOR: Maruyama T; Kasuga A; Ozawa Y; Nagata A; Abiko F; Suzuki Y; Saruta T
CORPORATE SOURCE: Department of Internal Medicine, Social Insurance Saitama Chuo Hospital, Japan.
SOURCE: Endocrine journal, (1997 Feb) Vol. 44, No. 1, pp. 43-51. Journal code: 9313485. ISSN: 0918-8959.
PUB. COUNTRY: Japan
DOCUMENT TYPE: (COMPARATIVE STUDY) Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199708
ENTRY DATE: Entered STN: 2 Sep 1997
Last Updated on STN: 2 Sep 1997
Entered Medline: 20 Aug 1997

L1 ANSWER 5 OF 9 USPATFULL on STN

TI Peptides, derivatives and analogs thereof, and methods of using same

AB Human proIslet Peptides (HIP) and HIP analogs and derivatives thereof, derived from or homologous in sequence to the human REG3A protein, chromosome 2p12, are able to induce islet neogenesis from endogenous pancreatic progenitor cells. Human proIslet Peptides are used either alone or in combination with other pharmaceuticals in the treatment of type 1 and type 2 diabetes and other pathologies related to aberrant glucose, carbohydrate, and/or lipid metabolism, insulin resistance, overweight, obesity, polycystic ovarian syndrome, eating disorders and the metabolic syndrome.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2007:101122 USPATFULL
TITLE: Peptides, derivatives and analogs thereof, and methods of using same
INVENTOR(S): Levetan, Claresa S., Bryn Mawr, PA, UNITED STATES
Upham, Loraine V., Mt. Laurel, NJ, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2007087971	A1	20070419
APPLICATION INFO.:	US 2006-441491	A1	20060525 (11)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2005-684819P	20050525 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: PEPPER HAMILTON LLP, ONE MELLON CENTER, 50TH FLOOR, 500 GRANT STREET, PITTSBURGH, PA, 15219, US
NUMBER OF CLAIMS: 24
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 7 Drawing Page(s)
LINE COUNT: 2989
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L1 ANSWER 6 OF 9 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
TI An examination of glutamate decarboxylase65 immunoreactive puncta with respect to rat ventral pallidum neurons after repeated cocaine administration.
AB The ventral pallidum is known to have topographically organized reciprocal γ-aminobutyric acid-ergic projections with the nucleus accumbens, and changes in these connections may play a role in mediating the behavioral sensitizing effect of repeated exposure to cocaine. The present study investigated glutamate decarboxylase-65 (GAD65) immunoreactivity in the rat ventral pallidum after repeated cocaine administration. Male Sprague-Dawley rats were administered bi-daily injections of 15 mg/kg cocaine or saline vehicle for 5 consecutive days. After 2 or 14 days of withdrawal, ventral pallidal sections were immunocytochemically processed for GAD65 immunoreactive puncta and counts were made. In both groups, there were no statistically significant differences in the number or density of GAD65 puncta in medial or lateral portions either in contact with neuronal cell bodies or in the neuropil after 2 or 14 days of withdrawal. The results suggest that there is no alteration in the number of GABAergic boutons expressing GAD65 immunoreactivity in the ventral pallidum after repeated exposure to cocaine. Copyright (C) 2000 Elsevier Science Ireland Ltd.
ACCESSION NUMBER: 2000128993 EMBASE
TITLE: An examination of glutamate decarboxylase65 immunoreactive puncta with respect to rat ventral pallidum neurons after repeated cocaine administration.
AUTHOR: De Leon K.R.; Todtenkopf M.S.; Stellar J.R.
CORPORATE SOURCE: K.R. De Leon, Department of Psychology, Northeastern University, 360 Huntington Avenue, Boston, MA 02115, United States. kdeleon@lynx.dac.neu.edu
SOURCE: Neuroscience Letters, (21 Apr 2000) Vol. 284, No. 1-2, pp. 69-72. .
Refs: 18
ISSN: 0304-3940 CODEN: NELED5
PUBLISHER IDENT.: S 0304-3940(00)00973-3
COUNTRY: Ireland
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical Biochemistry
030 Pharmacology
037 Drug Literature Index

040 Drug Dependence, Alcohol Abuse and Alcoholism
008 Neurology and Neurosurgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 21 Apr 2000
Last Updated on STN: 21 Apr 2000

L1 ANSWER 7 OF 9 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
TI Distribution of glutamate decarboxylase65 immunoreactive puncta on pyramidal and nonpyramidal neurons in hippocampus of schizophrenic brain.
AB Recent studies have reported an increase in GABA(A) receptor binding activity in several key corticolimbic regions, including the hippocampal formation, of postmortem schizophrenic brain. Because this change has been postulated to represent a compensatory upregulation of this receptor, the current report has sought to determine whether a decrease of glutamate decarboxylase (GAD), the enzyme responsible for the synthesis of GABA, may also be present in the hippocampus of schizophrenic subjects. A standard immunoperoxidase technique, together with a computer-assisted microscopic analysis, has been employed to evaluate the distribution of the 65 kDalton isoform of GAD (GAD65) in 12 normal controls and 13 schizophrenic subjects matched for age and postmortem interval (PMI). The results show no significant difference in the density of GAD65-immunoreactive (-IR) puncta in contact with pyramidal neurons (PN), nonpyramidal neurons (NP), or neuropil (NPL) in sectors CA(1-4) and their various sub-laminae. When the data were considered in relation to neuroleptic exposure, a significant positive correlation between the density of GAD65-IR puncta and drug dose was found on both PNs ($r = 0.814$, $P = 0.002$; $r = 0.777$, $P = 0.005$, respectively) and NPs ($r = 0.673$, $P = 0.023$; $r = 0.672$, $P = 0.024$, respectively) in sectors CA4 and CA3. A similar result was found in the stratum oriens of CA3 ($r = 0.704$, $P = 0.016$) and CA2 ($r = 0.774$, $P = 0.009$). In each instance, two neuroleptic free schizophrenics showed the lowest density of GAD65-IR puncta. There was no significant relationship between the density of GAD65-IR puncta with either age or PMI. Taken together with previous data showing an upregulation of GABA(A) receptor activity in sectors CA3 and CA2, particularly the stratum oriens, this study provides further evidence in support of the hypothesis that an intrinsic defect of GABAergic activity may occur in the hippocampal formation of schizophrenic patients and show dose-related increases in relation to neuroleptic exposure.
ACCESSION NUMBER: 1998238847 EMBASE
TITLE: Distribution of glutamate decarboxylase65 immunoreactive puncta on pyramidal and nonpyramidal neurons in hippocampus of schizophrenic brain.
AUTHOR: Todtenkopf M.S.; Benes F.M.
CORPORATE SOURCE: Dr. F.M. Benes, McLean Hospital, 115 Mill Street, Belmont, MA 02178, United States
SOURCE: Synapse, (1998) Vol. 29, No. 4, pp. 323-332. .
Refs: 37
ISSN: 0887-4476 CODEN: SYNAET
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 008 Neurology and Neurosurgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 14 Aug 1998
Last Updated on STN: 14 Aug 1998

L1 ANSWER 8 OF 9 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
TI Glutamic acid decarboxylase65 (GAD65) antibodies and insulin auto- antibodies in Japanese patients with noninsulin-dependent diabetes mellitus [3].

AB To clarify whether glutamic acid decarboxylase65 antibodies (GAD65 Ab) and insulin autoantibodies (IAA) are good predictive markers for insulin- dependency in NIDDM, we studied GAD65 Ab and IAA in NIDDM patients treated with diet alone or in combination with oral hypoglycemic agents. GAD65 Ab were found in 12 of 229 (5.2%, P=0.079 vs. control) NIDDM patients and IAA in 8 of 229 (3.5%). The frequency of GAD65 Ab and IAA positivity in NIDDM did not differ significantly from those of healthy controls (2/150, 1.3%, 2/150, 1.3%, respectively), but the frequency of patients who were positive for either GAD65 Ab or IAA, or both, was significantly higher than that of normal controls (17/229, 7.4% and 4/150, 2.7%, respectively, P<0.05). In addition, the prevalences of GAD65 Ab and of IAA in those patients whose disease durations, since the diagnosis of diabetes, were less than one year were significantly higher than those of controls (4/30, 13.3%, P<0.05, 4/30, 13.3%, P<0.05, respectively). We found no differences between GAD65 Ab positive- and negative-patients in either BMI or serum C-peptide levels. Over a one to five year follow-up period (mean 2.0yrs), serum C-peptide levels gradually decreased necessitating insulin treatment in three of the patients positive for GAD65 Ab and/or IAA (3/17, 17.6%; two were positive for both GAD65 Ab and IAA and one was positive for GAD65 Ab only). In contrast, only five patients negative for the two antibodies developed insulin requirement (5/212, 2.4%, P<0.01). These results suggest that GAD65 Ab and IAA are good markers for predicting the development of insulin dependency in NIDDM patients and that the predictive value for insulin-dependency in NIDDM is enhanced by measuring both antibodies.

ACCESSION NUMBER: 97105614 EMBASE
DOCUMENT NUMBER: 1997105614
TITLE: Glutamic acid decarboxylase65 (GAD65) antibodies and insulin auto- antibodies in Japanese patients with noninsulin-dependent diabetes mellitus [3].
AUTHOR: Maruyama T.; Kasuga A.; Ozawa Y.; Nagata A.; Abiko F.; Suzuki Y.; Saruta T.
CORPORATE SOURCE: Dr. T. Maruyama, Department of Internal Medicine, Social Insurance Saitama Chuo Hosp., 4-9-3 Kitaurawa, Urawa-shi, Saitama 336, Japan
SOURCE: Endocrine Journal, (1997) Vol. 44, No. 1, pp. 43-51. .
Refs: 30
ISSN: 0918-8959 CODEN: ENJOEO
COUNTRY: Japan
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 003 Endocrinology
006 Internal Medicine
026 Immunology, Serology and Transplantation
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 29 Apr 1997
Last Updated on STN: 29 Apr 1997

L1 ANSWER 9 OF 9 WPIDS COPYRIGHT 2007 THE THOMSON CORP on STN

TI Diagnosing and treating autoimmune diseases such as insulin-dependent diabetes mellitus and detecting antibodies to glutamin acid decarboxylase (GAD)65 in a sample, using GAD65 polypeptide
AN 2000-500251 [45] WPIDS
CR 1992-150489; 1992-425701; 1995-131360; 2000-095930
AB EP 1026238 A2 UPAB: 20050411
NOVELTY - Use of a glutamic acid decarboxylase65 (GAD65) polypeptide (I) or analog, chemical derivative, or pharmaceutically acceptable salt for diagnosing and treating autoimmune diseases such as insulin-dependent diabetes mellitus (IDDM), is new.
DETAILED DESCRIPTION - Use of a glutamic acid decarboxylase65 (GAD65) polypeptide (I) or analog, chemical derivative, or pharmaceutically acceptable salt for diagnosing and

treating autoimmune diseases such as insulin-dependent diabetes mellitus (IDDM), is new. (I) has the amino acid sequence X-Pro-Glu-Val-Lys-Y-Lys-Z - (II) where X is 1-10 amino acids, Y is Thr or Glu, and Z is 1-8 amino acids.

An INDEPENDENT CLAIM is also included for a pharmaceutical composition comprising labeled (I).

ACTIVITY - Immunosuppressive; antidiabetic. (I) was tested for the antidiabetic activity. The polypeptide derived from the GAD65 core sequence and from the homologous region of polio virus were compared. There was no significant difference in the proliferative activity of cultures of spleen lymphocytes exposed to either the polio or the GAD65 polypeptides. Both polypeptides stimulated a T-cell response which was higher than that found in the media control. The lack of difference in proliferation in the spleen cell population may be due to a lower frequency of GAD polypeptide specific T-cells. The islet infiltrating T lymphocyte (IITL) population, when evaluated in the same manner, showed a marked difference in cell proliferation. In this system, the response to the GAD65 polypeptide was 9-fold greater than that of either the culture media or the polio polypeptide. The data strongly suggests that the GAD65 is an important antigen for T-cell responses in the IITL population. This data suggests the molecular mimicry plays a role in the pathogenesis of diabetes.

MECHANISM OF ACTION - beta-cell destruction inhibitor.

USE - (I) is useful for diagnosing IDDM and for the preparation of medicament for treating IDDM or stiff man syndrome, detecting antibodies preferably autoantibodies to GAD65 in a sample by measuring GAD enzymatic activity utilizing monoclonal antibody specific to (I) bound to a lectin preferably ricin, where the polypeptide is employed in a competitive or non-competitive immunoassay, preferably radio immunoassay, sandwich immunometric assay or western blot assay in a direct of indirect format, classifying patients with autoimmune diseases such as IDDM, screening drugs that alters GAD function, generation of an antibody preferably monoclonal or polyclonal autoantibodies, blocking cellular autoimmune response, blocking recognition by a specific T-cell receptor or an major histocompatibility complex (MHC) receptor presenting an autoimmune antigen on the surface of an antigen presenting cell, stimulating a T-suppressor cell population, and competing for recognition of self-antigens at a level of antigen presentation (all claimed).

ADVANTAGE - A ready source of eukaryotic GAD65 polypeptide corresponding to the purified from the natural sources, while avoiding the problems associated with the isolation of naturally occurring eukaryotic non-GAD65 polypeptides when separating it from other eukaryotic non-GAD65 polypeptides. The absence of other eukaryotic GAD65 polypeptides is significant in that it allows the development of test systems which will only detect antibodies specifically reactive with GAD65 polypeptides. Providing eukaryotic GAD65 polypeptide in host cells has made possible to obtain much larger quantities of the polypeptide than are currently practicably available from natural sources. As a consequence, not only is it possible to use the polypeptide of the invention to more accurately classify patients with autoimmune diseases such as IDDM, but is also not possible to provide commercially useful quantities of GAD65 polypeptide for use in diagnostic system.

ACCESSION NUMBER:	2000-500251 [45] WPIDS
CROSS REFERENCE:	1992-150489; 1992-425701; 1995-131360; 2000-095930
DOC. NO. CPI:	C2000-150287 [45]
DOC. NO. NON-CPI:	N2000-370808 [45]
TITLE:	Diagnosing and treating autoimmune diseases such as insulin-dependent diabetes mellitus and detecting antibodies to glutamin acid decarboxylase (GAD)65 in a sample, using GAD65 polypeptide
DERWENT CLASS:	B04; D16; S03
INVENTOR:	CLARE-SALZLER M J; ERLANDER M G; KAUFMAN D L; TOBIN A J; ERLANDER M G R W J
PATENT ASSIGNEE:	(REGC-C) UNIV CALIFORNIA

COUNTRY COUNT: 16

PATENT INFO ABBR.:

PATENT NO	KIND	DATE	WEEK	LA	PG	MAIN IPC
EP 1026238	A2	20000809	(200045)*	EN	32	[7]
EP 1026238	B1	20070228	(200718)	EN		
DE 69233685	E	20070412	(200726)	DE		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
EP 1026238 A2	Div Ex	EP 1992-110308	19920617
EP 1026238 A2		EP 2000-102229	19920617
DE 69233685 E		DE 1992-633685	19920617
DE 69233685 E		EP 2000-102229	19920617

FILING DETAILS:

PATENT NO	KIND		PATENT NO	
EP 1026238	A2	Div ex	EP 519469	A
DE 69233685	E	Based on	EP 1026238	A

PRIORITY APPLN. INFO: US 1991-716909 19910618

```
=> s (GAD67)
L2        1767 (GAD67)

=> s l2 and (l1
UNMATCHED LEFT PARENTHESIS 'AND (L6'
The number of right parentheses in a query must be equal to the
number of left parentheses.

=> d his

     (FILE 'HOME' ENTERED AT 16:54:38 ON 22 SEP 2007)

     FILE 'MEDLINE, USPATFULL, DGENE, EMBASE, WPIDS' ENTERED AT 16:55:44 ON 22
     SEP 2007
L1             9 S (DECARBOXYLASE65)
L2          1767 S (GAD67)

=> s l1 and l2
L3             0 L1 AND L2

=> e tobin, a/au
E1             1     TOBIN Y M/AU
E2             1     TOBIN YVONNE M/AU
E3             0 --> TOBIN, A/AU
E4             2     TOBINA/AU
E5             2     TOBINA H/AU
E6             1     TOBINA T S/AU
E7           271     TOBINAGA/AU
E8             1     TOBINAGA E/AU
E9            80     TOBINAGA H/AU
E10            7     TOBINAGA I/AU
E11           18     TOBINAGA J/AU
E12            2     TOBINAGA J I/AU
```

Refine Search

Search Results -

Terms	Documents
L7 and (GAD)	1

Database:
US Pre-Grant Publication Full-Text Database
US Patents Full-Text Database
US OCR Full-Text Database
EPO Abstracts Database
JPO Abstracts Database
Derwent World Patents Index
IBM Technical Disclosure Bulletins

Search: L8

Refine Search | Recall Text | Clear | Interrupt

Search History

DATE: Saturday, September 22, 2007 Purge Queries Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=PGPB; PLUR=YES; OP=OR			
L8	L7 and (GAD)	1	L8
L7	tobin.in.	531	L7
L6	L1 and (GAD67)	0	L6
L5	L1 and (GAD)	1	L5
L4	L1 and (decarboxylase)	1	L4
L3	L1 and (GAD65)	0	L3
L2	L1 and (not GAD67)	1	L2
L1	20050164342	1	L1

END OF SEARCH HISTORY

Hit List

First Hit | Clear | Generate Collection | Print | Fwd Refs | Bkwd Refs | Generate OACS

Search Results - Record(s) 1 through 1 of 1 returned.

☐ 1. Document ID: US 20050164342 A1

L8: Entry 1 of 1 File: PGPB Jul 28, 2005

PGPUB-DOCUMENT-NUMBER: 20050164342
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20050164342 A1

TITLE: Cloned glutamic acid decarboxylase

PUBLICATION-DATE: July 28, 2005

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY
Tobin, Allan J.	Los Angeles	CA	US
Erlander, Mark G.	Tarzana	CA	US
Kaufman, Daniel L.	Santa Monica	CA	US

US-CL-CURRENT: 435/69.1; 435/232, 435/320.1, 435/325, 530/388.26, 536/23.2

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw D

Clear | Generate Collection | Print | Fwd Refs | Bkwd Refs | Generate OACS

Terms	Documents
L7 and (GAD)	1

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